IN THE SPECIFICATION

Please amend paragraph [0192] as indicated below.

[0192] After an MI injury occurs macrophages tend to infiltrate the infarct region. The macrophages release matrix metalloproteinases (MMPs). As members of a zinc-containing endoproteinase family, the MMPs have structural similarities but each enzyme has a different substrate specificity, produced by different cells and have different inducibilities. These enzymes cause destruction in the infarct zone. One important structural component destroyed by MMPs is the extracellular matrix (ECM). The ECM is a complex structural entity surrounding and supporting cells that are found within mammalian tissues. The ECM is often referred to as the connective tissue. The ECM is composed of 3 major classes of biomolecules; structural proteins: for example collagen and elastin, specialized proteins for example fibrillin, fibronectin, and laminin, and proteoglycans: these are composed of a protein core to which is attached long chains of repeating disaccharide units termed of glycosaminoglycans (GAGs) forming extremely complex high molecular weight components of the ECM. Collagen is the principal component of the ECM and MMP induce ECM degradation and affect collagen deposition. Inhibitors of MMP(s) exist 1970 and some of these inhibitors are tissue specific. It was previously demonstrated that acute pharmacological inhibition of MMPs or in some cases a deficiency in MMP-9 that the left ventricle dilatation is attenuated in the infarct heart of a mouse ~~(Creemers, E. et.al. "Matrix Metalloproteinase Inhibition After Myocardial Infarction" A New Approach to Prevent Heart Failure? Circ Res. Vol 89 No. 5, 2315-2326, 1994)~~ (Creemers, E. et al. "Matrix Metalloproteinase Inhibition After Myocardial Infarction: A New Approach to Prevent Heart Failure?" Circ. Res. Vol. 89:201-210 (2001)). The inhibitors of MMPs are referred to as tissue inhibitors of metalloproteinases (TIMPs). Synthetic forms of MMPIs also exist for example BB-94, AG3340, Ro32-355b and GM 6001. It was previously shown that MMPIs reduce the remodeling in the left ventricle by reducing wall thinning. These experiments were performed on rabbits. In addition, this study also demonstrated that MMPI increases rather than decreases neovascularization in the subendocardium ~~(Lindsey et. al. "Selective matrix metalloproteinase inhibitors reduce left ventricle remodeling but does~~

~~not inhibit angiogenesis after myocardial infarction," Circulation 2002 Feb. 12;105 (6):753-8)~~ (Lindsey, M. et al. "Selective Matrix Metalloproteinase Inhibition Reduces Left Ventricular Remodeling but does not Inhibit Angiogenesis after Myocardial Infarction," Circulation 105(6):753-758, (Feb 2002)). In the one embodiment MMPIs may be introduced to the infarct region to delay the remodeling process by reducing the migration of fibroblasts and deposition of collagen and prevent ECM degradation, reduce leukocyte influx and also reduce wall stress. In one embodiment, the MMPIs may include the following TIMPs including but not limited to TIMP-1, TIMP-2, TIMP-3 and TIMP-4 introduced to the infarct region in combination with introducing any of the described structurally reinforcing agents to the infarct region. In another embodiment, naturally occurring inhibitors of MMPs may be increased by exogenous administration of recombinant TIMPs. In another embodiment, the MMPI comprises a synthetically derived MMPI introduced to the infarct region in combination with introducing any of the described structurally reinforcing agents and/or applied stimulating devices (eg. PG) to the infarct region. The introduction of MMPIs to the infarct zone may be accomplished by several different methods. It is critical that the introduction of these MMPI agents be accomplished by a minimally invasive technique. In one embodiment, MMPI agents will be introduced to the region by a minimally invasive procedure to prevent ECM degradation. An agent or dispersion will be introduced in one embodiment by multiple injections to the infarct region. This results in prevention of ECM degradation and increased strength to the regional wall. In one embodiment, the MMPI agent may be injected in to the infarct zone during an open chest procedure via a minimally invasive procedure. In another, the minimally invasive procedure may include one of sub-xiphoid and percutaneously. In another embodiment, the percutaneous introduction into the infarct zone may include one of intra-ventricular needle, transvascular needle catheter and retrograde venous perfusion. In addition, the MMPI agents may be introduced via suspension or sustained release formula for example introduced in microparticles.